

IN THE CLAIMS

Please amend the Claims as follows:

1. (original) Method of identifying, selecting and/or characterizing a compound which modulates the activity of at least one Src family kinase, comprising the steps of:
 - a. cultivating at least one cell (or at least one cell line) containing at least one nucleic acid coding for a Src family kinase or a mutated Src family kinase under suitable conditions,
 - b. expressing the nucleic acid(s) coding for a Src family kinase or a mutated Src family kinase in the cell(s) of step (a) under suitable conditions,
 - c. contacting the cell(s) of step (b) with at least one test compound and
 - d. determining whether the phenotype of the cell(s) of step (c) is changed as compared with the phenotype of the cell(s) of step (b), whereby a difference in the phenotype indicates that said test compound modulates the activity of at least one Src family kinase.
2. (original) Method of claim 1, wherein the nucleic acid coding for Src family kinase or a Src family kinase mutant is inducibly expressed according to step (b).
3. (currently amended) Method of claim 1 ~~or claim 2~~, wherein at least one cell (or at least one cell line) expresses a wildtype (wt) Src family kinase.
4. (currently amended) Method of ~~any of claim[[s]] 1 to 3~~, wherein at least one cell (or at least one cell line) expresses a Src kinase family member mutant, which displays a hyperactive form of a Src family kinase.

5. (original) Method of claim 4, wherein at least one cell (or at least one cell line) expresses a Src kinase family member mutant, which contains at the regulatory tyrosine residue (phosphorylation site) a mutation, which does not allow the tyrosine residue to be phosphorylated, preferably by deletion, substitution or modification of the regulatory tyrosine residue.
6. (currently amended) Method of ~~any of~~ claim[[s]] 1 to 5, wherein at least one cell (or at least one cell line) expresses a Src kinase family member mutant, which displays a “kinase dead” form of a Src family kinase.
7. (original) Method of claim 6, wherein at least one cell (or at least one cell line) expresses a Src kinase family member mutant, which contains a mutation at the ATP binding site of a Src family kinase, which does not allow to bind ATP properly, preferably by deletion, substitution or modification of at least one residue of the ATP binding site.
8. (currently amended) Method of ~~any of~~ claim[[s]] 1 to 7, wherein at least one cell (or at least one cell line) expresses a Src kinase family member mutant, which displays a “specificity determining” form of a Src family kinase.
9. (original) Method of claim 8, wherein at least one cell (or at least one cell line) expresses a Src kinase family member mutant, which contains a mutation of at least one residue, which is characteristic for the Src kinase family, preferably at least one residue in the ATP binding site of a Src family kinase, which allows to identify test compounds which do not bind to the Src family mutant (in contrast to wt Src).

10. (currently amended) Method of ~~any of~~ claim[[s]] 1 ~~to~~ 9, wherein at least one cell (or at least one cell line) expresses a Src kinase family member “double” mutant, which contains (i) at least one mutation leading to a hyperactivated form of a Src family kinase and (ii) at least one mutation at a specificity determining region.
11. (currently amended) Method of ~~any of~~ claim[[s]] 1 ~~to~~ 10, wherein the mode of action (direct or indirect), the specificity, and/or the effectiveness of a test compound on the (activity of a) Src family kinase is/are determined.
12. (currently amended) Method of ~~any of~~ claim[[s]] 1 ~~to~~ 11, wherein at least one, preferably two, three or four, nucleic acids coding for a mutated Src family kinase are selected from the group consisting of SEQ ID NOs 1 to 4 (Src kinase mutant sequences), SEQ ID NOs 5 to 8 (Yes kinase mutant sequences), SEQ ID NOs 9 to 12 (Fgr kinase mutant sequences), SEQ ID NOs 13 to 16 (Fyn kinase mutant sequences), SEQ ID NOs 17 to 20 (Lck kinase mutant sequences), SEQ ID NOs 21 to 24 (Hck kinase mutant sequences), SEQ ID NOs 25 to 28 (Lyn kinase mutant sequences), SEQ ID NOs 29 to 32 (Blk kinase mutant sequences), and SEQ ID NOs 33 to 36 (Yrk kinase mutant sequences).
13. (currently amended) Method of ~~any of~~ claim[[s]] 1 ~~to~~ 12, wherein the method is based on four cells (or four cell lines), each of them containing one of the four nucleic acids, each of them expressing one of the mutant sequences of one Src family member according to claim 12, e.g. SEQ ID NOs 1 to 4 for Src.
14. (currently amended) Method of ~~any of~~ claim[[s]] 1 ~~to~~ 13, wherein the cell or cell line is transfected with a vector, in particular an expression vector, containing the nucleic acid of a Src family kinase or a Src family kinase mutant.

15. (original) Method of claim 14, wherein the vector is selected from the group consisting of pcDNA4/TO, PcDNA3-derivatives, and pcDNA5/TO.
16. (currently amended) Method of ~~any of~~ claim[[s]] 1 to 15, wherein the expression of the nucleic acid in the transfected cell (or cell line) is induced by adding an inductor, preferably an antibiotic, e.g. tetracycline.
17. (currently amended) Method of ~~any of~~ claim[[s]] 1 to 16, wherein the induced expression causes an overexpression of the nucleic acid in the transfected cell or cell line.
18. (currently amended) Method of ~~any of~~ claim[[s]] 1 to 17, wherein the transfected cell is an eukaryotic cell, preferably a vertebrate cell, more preferably a mammalian cell, most preferably a human cell.
19. (original) Method of claim 18, wherein the cell is an immortalised cell, a tumor cell line or a lymphoid cell.
20. (currently amended) Method of ~~any of~~ claim[[s]] 1 to 19, wherein the phenotype change of the transfected cells upon induction and addition of a test compound is determined by qualitative and/or quantitative analysis, preferably by eye, microscope or image analysis.
21. (currently amended) Method of ~~any of~~ claim 20, wherein the phenotype change is determined by assessing whether the cellular phenotype of step (c) is changed as compared with the cellular phenotype of step (b), whereby a difference in the cellular phenotype indicates that said test compound modulates the activity of a Src family kinase.

22. (currently amended) Compound identified, selected and/or characterized by a method of ~~any~~ of claim[[s]] 1 ~~to 21~~.

23. (original) Compound of claim 22 as a medicament, particularly for the treatment of diseases, which are at least in part caused by a Src family kinase.

24. (currently amended) ~~Use of a compound of claims 22 or 23 for the production of a medicament~~ A method for the treatment of diseases, which are at least in part caused by a Src family kinase, particularly by a dysfunction of a Src family kinase, in particular cancer, hypercalcemia, restenosis, osteoporosis, osteoarthritis, symptomatic treatment of bone metastasis, rheumatoid arthritis, inflammatory bowel disease, multiple sclerosis, psoriasis, lupus, graft vs. host disease, T-cell mediated hypersensitivity disease, Hashimoto's thyroiditis, Guillain-Barre syndrome, chronic obstructive pulmonary disorder, contact dermatitis, cancer, Paget's disease, asthma, ischemic or reperfusion injury, allergic disease, atopic dermatitis, transplant rejection or allergic rhinitis[[.]], which method comprises administering a medicament comprising a compound of claim 22, in an amount effective for said treatment.

25. (currently amended) Pharmaceutical composition containing a compound according to claim[[s]] 22 ~~or 23~~, and a pharmaceutically acceptable carrier, adjuvant or vehicle.

26. (original) Pharmaceutical composition according to claim 25 for the preparation of a medicament for the treatment of cancer, hypercalcemia, restenosis, osteoporosis, osteoarthritis, symptomatic treatment of bone metastasis, rheumatoid arthritis, inflammatory bowel disease, multiple sclerosis, psoriasis, lupus, graft vs. host disease, T-cell mediated

hypersensitivity disease, Hashimoto's thyroiditis, Guillain-Barre syndrome, chronic obstructive pulmonary disorder, contact dermatitis, cancer, Paget's disease, asthma, ischemic or reperfusion injury, allergic disease, atopic dermatitis, transplant rejection or allergic rhinitis.

27. (currently amended) ~~Use of a pharmaceutical composition of any of claims 25 or 26 for the production of a medicament~~ A method for the treatment of diseases, which are at least in part caused by a Src family kinase, preferably cancer, hypercalcemia, restenosis, osteoporosis, osteoarthritis, symptomatic treatment of bone metastasis, rheumatoid arthritis, inflammatory bowel disease, multiple sclerosis, psoriasis, lupus, graft vs. host disease, T-cell mediated hypersensitivity disease, Hashimoto's thyroiditis, Guillain-Barre syndrome, chronic obstructive pulmonary disorder, contact dermatitis, cancer, Paget's disease, asthma, ischemic or reperfusion injury, allergic disease, atopic dermatitis, transplant rejection or allergic rhinitis[.], which method comprises administering an effective amount of a medicament comprising a pharmaceutical composition of claim 25.